filed May 21, 1997, which is a Continuation-In-Part of Application Serial No. 08/858,660 filed May 19, 1997, now abandoned, which is based on Provisional Application Serial No. 60/017,157, filed May 20, 1996, and Provisional Application Serial No. 60/020,043 filed June 19, 1996. Applicants claim the priority of these Application under 35 U.S.C. §§120 and 119(e). The disclosures of the Applications having the Serial Nos. 08/861,105, 08/858,660 and 60/017,157 are hereby incorporated by reference in their entireties. - -

On Page 10, line 23, after "conformational" please replace "isotope" with - - epitope - - , therefor.

On Page 14, line 20 after "FIGURE" please replace "1 shows" with -- 1A-1B show -- therefor;

on line 22 after "Envs" please insert -- (Fig. 1B) -- therefor, and after "Env" please insert -- (Fig. 1A) -- therefor;

on line 26 after "FIGURE" please replace "2 shows" with -- 2A-2C show -- therefor; and

on line 32 after "Figure" please replace "2a," with -- 2A - - therefor.

On Page 15, line 3, after "Figure" please replace "2b," with -- 2B - - therefor;

on line 4 after "as in" please replace "2a," with -- Figure 2A - - therefor;

on line 5 please replace "2c is the same as 2a" with - - Figure 2C is the same as Figure 2A - - therefor;

on line 8 after "FIGURES" please replace "3a-3d" with -- 3A-3G -- therefor;

on line 11 please replace "3a" with -- 3A -- therefor;

on line 15 after "Figure" please replace "3b shows" with -- 3B-3E show -- therefor;

on line 22 after "Figure" please replace "3c" with -- 3F -- therefor, and after "HOS.CD4 cells (human osteosarcoma," please insert - - which has been deposited with the American Type Culture Collection, Rockville Md., 20852 on May 25, 2000 under the Budapest Treaty - -;

on line 23 after "Figure" please replace "3d" with -- 3G -- therefor;

and

reactions. First strand cDNA was primed with oligo-dT using Superscript reverse transcriptase as per manufacturer's direction (Gibco/BRL) and products were amplified with primers hybridizing to the 5' and 3' untranslated regions of CC-CKR-5 (upstream CTCGGATCCGGTGGAACAAGATGGATTAT (SEQ ID NO: 1); downstream CTCGTCGACATGTGCACAACTCTGACTG (SEQ ID NO: 2)) or to glyceraldehyde-3-phosphate dehydrogenase using a Taq/Pwo polymerase mixture (Boehringer Mannheim). To control for the presence of genomic DNA, control cDNA reactions in which reverse transcriptase was omitted were prepared in parallel. These were uniformly negative. To test the linearity of amplification, a ten-fold dilution series (lanes 1-5) starting at 1 pg of pcCKR5 plasmid DNA was amplified under conditions identical to those above. In lane 6, no DNA was added. Monocytes were prepared by overnight adherence to plastic. T cells were prepared from the monocyte-depleted preparation by adherence to anti-CD2-coated beads (Dynal).

Please substitute the following amended paragraph for the original paragraph starting on page 48, lines 28-34, continuing on to page 49, lines 1-13:

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Murine cells transfected with human CD4 are resistant to infection with all tested strains of HIV. To determine whether chemokine receptors could confer susceptibility to infection, the different receptor genes are stably introduced into murine 3T3.CD4 cells. Cells expressing CC-CKR-1 (CCR1), CC-CKR-2B (CCR2B), CC-CKR-3 (CCR3), CC-CKR-4 (CCR4), Duffy, or ~~fusion~~ fusin (CXCR4) are all resistant to infection with HIV-luciferase pseudotyped with macrophage-tropic Envs, but are infected with virus bearing amphotropic Env (Fig. ~~3a~~ 3a-3c). Expression of CC-CKR-5 permitted infection with the macrophage-tropic pseudotypes, but these cells are resistant to infection mediated by HXB2 Env (Fig. ~~3a~~ 3a-3c). Only ~~fusion~~ fusin-expressing 3T3.CD4 cells are permissive for infection with this T-tropic virus (Fig. ~~3a~~ 3a-3c). The chemokine receptors are expressed on the surface of the 3T3.CD4 cells, as assessed by mobilization of intracellular free Ca^{++} in response to the appropriate chemokines (Fig. ~~3b~~ 3d-3g). Cells expressing CC-CKR-5 (CCR5) responded to RANTES, MIP-1α and MIP-1β, consistent with known β-chemokine reactivities. Infection of the 3T3.CD4 cells expressing CC-CKR-5 with macrophage-tropic virus is blocked by a mixture of the three chemokines that efficiently

activate this receptor as well as by anti-CD4 antibody (Fig. ~~3a~~ 3a-3c). Infection of the fusion-expressing cells with T-tropic virus is also blocked by anti-CD4, but is completely refractory to treatment with chemokines. Thus, these results suggest that only CC-CKR-5 mediates entry of macrophage-tropic Envs, that T-tropic envelope glycoproteins do not use this co-receptor for entry, and that β-chemokines block entry of the macrophage-tropic virus by specifically binding to this receptor.

19 Please substitute the following amended paragraph for the original paragraph on page 49, lines 15-28:

09/21/06

Stable expression of CC-CKR-5, but not of the other β-chemokine receptors, in human HOS.CD4, HeLa.CD4, and U87MG.CD4 cells also conferred upon these cells susceptibility to infection with macrophage-tropic HIV-1 (Fig. ~~3c, 3d~~ 3h, 3i-3j and data not shown). As observed in the transient transfections, stable co-expression of both CC-CKR-5 and CD4 is required for viral entry into the HeLa cells (Fig. ~~3d~~ 3i-3j). Infection of these cells with macrophage-tropic virus is reduced by 70-80% upon treatment with a mixture of chemokines (Fig. ~~3d~~ 3i-3j). High levels of β-chemokines failed to inhibit infection of HOS.CD4 cells. In general, inhibition with β-chemokines is consistently less efficient in the non-lymphoid cells expressing CD4 and CC-CKR-5 than in the PM1 cells.